AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 102:295–299 (1997)

Notes and Comments

Comment on "Hypoplastic Area Method for Analyzing Enamel Hypoplasia" B.E. Ensor and J.D. Irish, *American Journal of Physical Anthropology* (1995) 98:507–517.

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The article by Ensor and Irish (1995) attributes to itself the first "methodology to determine the amount [duration] of hypoplasia for comparing populations based on individuals," and states that "no specific method for assessing the level of stress for comparative purposes has appeared in the literature" (p. 507). This assertion is untrue. Their characterization of the work, which, in fact, broadly disseminated the first such methodology (Blakey and Armelagos, 1985) as one that, "assumes uniformity between all defects and does not account for the range of variability in the duration of stress" (p. 507) patently misrepresents our method.

Blakey and Armelagos (1985) utilize a method first developed by Blakey (1981) that involves measurements of both the incisal (onset) and cervical (cessation) aspects of human dental enamel hypoplasia. Many others have utilized aspects of this method. It analyses and graphically estimates duration, as well as the ages when hypoplastic episodes begin and end in each individual under study. So well was this new methodology appreciated by the authors, that the term "methodology" is listed as a Key Word in that publication. The first sentence of the abstract reads, "The month of onset, duration, and incidence of dental enamel hypoplasia and hypocalcification was determined . . ." (p. 371). The y-axis of one of the publication's charts (p. 377) is labelled "Duration" for those who may not attend to the abstract and text.

Blakey's method more truly represents stress duration in *individuals* than does this recent study, because hypoplasia in different teeth, whose periods of development overlap, are evaluated as representing a continuous episode for those individuals. Ensor and Irish base their durations on the sum of means from the crowns of tooth types considered in isolation. Their method permits the error of counting twice the duration of hypoplasia on different teeth, whose development is simultaneous.

Blakey's method, uniquely, provides data on the specific ages during which stress chronicity occurred. This method is well suited for population comparisons of the durations of hypoplasia and hypocalcification, although we no longer suggest its use for determining age at onset and cessation for hypocalcification, specifically (see Blakey et al., 1994).

Surely, Ensor and Irish do not mean to suggest that their use of tests of statistical significance constitutes a new method.

When one re-invents the wheel, one is less likely to create a better one than when one builds upon previous invention. Those of us who have published extensively on these dental indicators, know that longitudinal and experimental studies are most needed in order to test the assumptions of chronologic methods. Blakey and Armelagos (1985, pp. 373–375) make that point clearly in connection with the differing developmental assumptions of tooth-specific and population-specific chronologies (a crucial methodological issue that is also raised in our work for the first time).

Most importantly, Ensor and Irish (1995) indicate that they were knowledgeable of our work (p. 505). They would, in any event, have found frequent reference to its meth-

Received 4 January 1996; Accepted 6 May 1996.

ods in the literature (see for example the references to our study in most of the methodology chapters of Goodman and Capasso, 1992), yet they describe it as unrelated to duration methodology. One of the most important reviews of dental defect literature (Goodman and Rose, 1990, p. 92) references our 1985 article as measuring defect width, intended to measure duration. Hutchinson and Larsen (1988), whose population comparisons of hypoplasia chronicity are a focal point of Ensor and Irish's criticism, also cite our study as a method for duration estimation that informed their method and analysis (Larsen and Hutchinson, 1992, pp. 163–164). It seems unreasonable to assume that Bradley Ensor and Joel Irish were unfamiliar with our prior development of a measurement of dental defect "area" or chronicity that can be used for population comparisons.

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Reply to Blakey and Armelagos, With Additional Remarks on the Hypoplastic Area Method

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In their appraisal of our Hypoplastic Area method article (Ensor and Irish, 1995a), Blakey and Armelagos (1996) list a variety of criticisms, primarily regarding our reinventing the wheel. We find the majority of these criticisms to be both interesting and a bit disconcerting—yet not convincing. We address each criticism on a point-by-point basis.

In their first sentence, Blakey and Armelagos take not one, but two quotes in our original paper (Ensor and Irish, 1995a) out of context. These misquotes result in a distortion of our intended meaning. First, we do not claim to have the "*first* 'methodology to determine the amount [duration] of hypoplasia for comparing populations based on individuals' " (Blakey and Armelagos, 1996). The actual statement reads. ". . . an *easy-to-use* methodology to determine the amount of hypoplasia for comparing populations based on individuals" (Ensor and Irish, 1995a, p. 507; emphasis added). Their second misquote reads ' "no specific method for assessing the level of stress for comparative purposes has appeared in the literature' " (Blakey and Armelagos, 1996). Our actual statement is ". . . no specific method for assessing the level of stress for comparative

Received 20 March 1996; Accepted 6 May 1996.

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purposes has appeared in the literature (*except for Hutchinson and Larsen, 1988; see later*)" (Ensor and Irish, 1995a, p. 507; emphasis added). Thus, we acknowledged a previous, pioneering study that measures the amount/area of enamel hypoplasia (EH) in individuals, and compares the means for each tooth type using t-tests.

However, there are important differences between the Hutchinson and Larsen method and our's. Their method is applicable when comparing one tooth type at a time. Our's ". . . uses information from several tooth types, which can be used in the same manner [as the Hutchinson and Larsen method]; it [Hypoplastic Area] can also be used to acquire a value for an individual dentition" (Ensor and Irish, 1995a, p. 509). These differences prompted us to write that "To our knowledge, no similar methodology [Hypoplastic Area] has been proposed to compare individuals quantitatively" (Ensor and Irish, 1995a, p. 507). Our method is discussed in greater detail below.

Blakey and Armelagos (1996) also maintain that we misrepresented their method by characterizing it as one that "assumes uniformity between all defects and does not account for the range of variability in the duration of stress" (Ensor and Irish, 1995a, p. 507). In this criticism, they are absolutely correct. We inadvertently referenced their 1985 paper, on the duration of deciduous enamel defects, in a list of studies associated with analyzing frequencies of the numbers of hypoplastic events. We intended to reference their work further down in the same paragraph—in a sentence about advancements in analysis of age distributions of hypoplasia. For this mistake, we apologize.

Next, Blakey and Armelagos (1996) present a cursory description of the Blakey method (Blakey, 1981; Blakey and Armelagos, 1985), compare it to our Hypoplastic Area method, and list its perceived advantages. As we will demonstrate, there are numerous differences which preclude such a direct comparison. The most obvious difference is that the two methods measure different aspects of EH: individual chronicity and stress duration (theirs) vs. specific tooth and/or individual amount/area (ours).

Their method first entails the measurement of EH lesions on dental crowns using a sliding caliper. In their 1985 study, Blakey and Armelagos measured all available deciduous teeth in a sample of 50 sub-adults. For acute lesions (those arbitrarily set at $\leq$1.5 mm in width), measurements are taken from the cervical line to the center of the defect. For chronic EH ($>$1.5 mm), measurements are taken from the cervical line to the incisal (onset) and cervical (cessation) aspects of the lesion (Blakey and Armelagos, 1985, 1996). We use an analogous technique to record the total amount (area) of EH on crowns. However, as noted by Blakey and Armelagos (1996), "many others have utilized aspects of this method." Indeed, measurement of EH defects in this manner is often standard, accepted procedure.

Blakey and Armelagos (1985) treat the duration of chronic defects as the number of monthly developmental segments affected, subtracted from the number of months for a crown's development. They record episodes spanning $\leq$1.5 mm in width as equal to 2 months or less in duration. This procedure, then, involves determining the width of monthly growth segments for each tooth analyzed. Because their method is oriented toward age of occurrence of defects, their duration estimates must consider the overlap of developmental monthly segments between teeth. When defects on different teeth overlap in developmental periods, the duration is estimated by the continuous span of defects on these teeth.

As they note (Blakey and Armelagos, 1985), a major problem in developing their duration methodology involved choosing whether timing should be based on 1-month enamel segments derived from crown height averages for tooth types in the sample, or on crown heights of individual teeth. They chose to use individual crown heights. However, they acknowledge that "either method may only be possible with deciduous dentition where occlusal attrition is minimal and crown height can be accurately measured" (Blakey and Armelagos, 1985, p. 375).

The Hypoplastic Area method begins by deriving a cervical two-thirds crown height estimate, which serves as the cut-off for analysis to avoid the problem of occlusal attrition in permanent teeth (see Ensor and Irish, 1995a, for a detailed discussion). In addition, we measure only the polar teeth in

each of the eight morphogenetic fields, based on the observations by Goodman and Armelagos (1985) that they are more susceptible to EH. Acute defects (bands ≤0.5 mm) are given a value of .10. For each chronic lesion, the percentage of the crown that is affected is recorded (e.g., a tooth with 60% of its crown affected receives a value of .60). When the total amount of hypoplasia (both acute and chronic) for a tooth is summed, the value is termed the Tooth Hypoplastic Area (THA). Thus, a tooth with two incidences of acute hypoplasia and a chronic lesion occupying 60% of the remaining crown would receive a THA value of .80.

The THA is the basic score for a tooth, and it should be apparent that it is *not* an indicator of age of onset and termination (duration). Instead, it is a score for the total *amount* of hypoplasia covering the crown. In other words, our method is concerned with how much childhood stress (as indicated by total EH in the polar teeth) the individual experienced. Any references made to duration are only in the context of differentiating between types of defects—acute vs. chronic—for the purpose of categorization and scoring. If our aim was to measure chronicity, we would have named the method something akin to "Hypoplastic *Duration*."

Our THA scores are most comparable to values achieved by the Hutchinson and Larsen (1988) method, which utilizes millimeters of vertical hypoplasia for an indicator of level of stress. However, as noted, our method goes farther—through the calculation of the Individual Hypoplastic Area (IHA). The IHA is the mean of the eight THA values, and results in a single score for an individual.

The THA and IHA scores are interval-level variables that are suitable for use in univariate, and particularly multivariate statistical applications. For example, through the use of discriminant analysis, individuals from the three samples in our study (Ensor and Irish, 1995a) were classified based on differences in EH amount/area. As we commented, the actual univariate or multivariate techniques employed on the THA and IHA values are up to the individual researcher (Ensor and Irish, 1995a). Clearly, however, the use of statistical tests of significance are not what differentiates the two methods.

Blakey and Armelagos (1996) go on to say that "Blakey's method more truly represents stress duration in *individuals* [their emphasis] than does this recent [Ensor and Irish, 1995a] study." They additionally claim that their method ". . . provides data on the specific ages during which stress chronicity occurred." "This method is well suited for population comparisons of the durations of hypoplasia . . ." We agree. On the other hand our method, as shown, is designed for individual and population comparisons based on: (1) the total amount or area of EH on individual teeth (THA), and/or (2) a single score for each individual using the IHA.

Therefore, we think it is obvious that we did not re-invent the wheel. From the outset, we emphasized that our 1995 article was oriented toward analyzing the amount of hypoplasia. We noted that analysis of EH age occurrence would have to be the topic of another investigation; the reason is that the Hypoplastic Area method is not designed for acquiring age of occurrence estimates. It is geared toward levels of stress. Indeed, it was originally termed the "Degree of Stress method" in our first draft. For us to determine chronological estimates of defects, those measurements taken to find EH amount/area must be used in a different manner. In a poster presentation at the Oakland AAPA meeting (Ensor and Irish, 1995b) we correlated level of stress with chronicity. For level of stress, we used the Hypoplastic Area method. For estimating the age of occurrence, we used the Blakey method, albeit with crown height averages, following Goodman and co-workers' (1980, 1984) approach to the permanent dentition based on developmental charts by Massler et al. (1941).

Finally, Blakey and Armelagos (1996) make one additional comment with which we disagree. They conclude that the Hutchinson and Larsen (1988) method of measuring the area of hypoplasia is a "focal point" of our criticism. This remark does not accurately represent our opinion of Hutchinson and Larsen's method, which we used as a reference point for our own work. In our paper we attempted to briefly explain the methodology and rationale behind their method, and compare and contrast it with our's. At no

time did we purposefully criticize the Hutchinson and Larsen method; we simply stated how our's differs from it and results in additional information.

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